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Canine Virus Forum

Members of the Forum

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Dr. W. MANSI, Veterinary Laboratory, Ministry of Agriculture.

Dr. R. F. MONTGOMERIE, Wellcome Research Laboratories.

Mr. C. E. WOODROW, Practising Veterinary Surgeon.

Capt. J. A. McBEE, Veterinary Surgeon, United States Air Force.

Question No. 1

(1a) *In the opinion of the forum is there any special risk attached to the injection of the egg-adapted vaccine into a dog, not previously immunised, who has recently been in contact with distemper or hard pad infection and who may be in the incubative or very early stages of the disease?*

(1b) *Clinical observation suggests that in cases of hard pad or distemper, the egg-adapted vaccine has more obvious curative effect and that the incidence of central nervous system involvement is lower if it is used in cases of illness of at least 14 days' duration rather than in the early case. Does the forum agree? And if so, what is its explanation?*

Dr. Montgomerie said that the first part of the question was of great interest; if he was to answer on laboratory experience, he would say there was no special risk in the injection of egg-adapted virus in the dog in the incubative stage of either distemper or hard pad disease. He recognised that experience in the laboratory was often not the experience in the field. There must have been a number of instances of the injection of egg-adapted virus to dogs when in the incubative stage of, say, hard pad. In a few cases dogs recently injected had gone down with hard pad disease. From these cases they had isolated virulent hard pad virus. They had searched most carefully for egg-adapted virus in the small number of cases of that type which they had had, and in no cases were they able to isolate virus on the chick embryo membrane in the laboratory. On the other hand, in a number of these cases they had isolated virulent hard pad type of virus. There was no means of saying "Yea or Nay" to the suggestion that they had isolated the egg-adapted virus, turned virulent. They had tried on a number of occasions to get the egg-adapted virus to mutate in the dog or ferret, and so far had not succeeded. As far as present experience was concerned, it had been very remarkable that trouble tended to appear in one kennel or in one group of dogs. In two of the cases they were able to get a good history from the owner and there was clear evidence of mild illness in the dogs about two weeks before the egg-adapted vaccine had been injected. He suggested that in these cases, hard pad virus had invaded the kennels and was present in the dogs at the time the egg-adapted virus was injected. Whether the disease would have occurred in any case or whether the egg-adapted virus precipitated the happening it was difficult to say. He was inclined to the opinion that the injection of the egg-adapted virus had precipitated the happening.

On the second part of the question as to whether egg-adapted vaccine had curative effects it was obvious that the curative effects were more in the later stages than in the early stages, but he had no good evidence to offer on that. It seemed to him that in the early incubation the injection of the egg-adapted vaccine was not a good therapeutic agent, but on the other hand it had a level of therapeutic value in developed cases.

Mr. Woodrow said that as to the effect of the injection of the vaccine to a dog which might be in the incubative stage, the injection did appear at times to "trigger off" the disease. In all instances where they had failures or a breakdown of the immunity, they had been cases of "shop" dogs. Although on clinical examination they might show no sign of infection, he thought they might have developed it in any case, whether injected with vaccine or not.

Capt. McBee said that in the case of a dog in contact with distemper infection he would not use a vaccine because he felt he might be getting into difficulty from its use. The vaccine could precipitate a position it would otherwise not have.

Dr. Larin said that in his experience of distemper virus, when the dog was in the incubative stage or early stages of the disease, an inoculation with the virulent virus did not change the course of the disease at all. He did not know whether the egg-adapted virus would behave in a different way. As to the curative effect of either virulent or egg-adapted virus, he could not conceive as to how the healing might occur, unless it was a sort of witchcraft.

Regarding the immunising value of egg-adapted virus, he said that his Station investigated many cases where egg-adapted virus produced no immunity at all. This was quite comprehensible because egg-adapted virus was prepared with one strain, and thus it could not produce immunity to all current virus strains. For this reason, it was difficult to expect it to work in 100 per cent. of the cases. It probably would not always work in even 50 per cent. if immunised dogs were exposed to a different type of the virus.

Dr. Mansi said that he could not see that it depended on whether it was in the early stages or the later stages. With distemper in the late stage there was no specific treatment whatever, and in giving vaccine they could not really make the dog worse; it might help, but he did not think there was anything specific in the way that it worked. In the early and incubative stages it was another problem. If they had doubts about a dog being exposed or in contact, he would suggest a serum would be much better at that particular stage.

Dr. Montgomerie said that taking virus of the hard pad type rather than the old-fashioned distemper, they had never succeeded in the laboratory in reproducing the field type of case. They tried to see whether the egg-adapted virus influenced the hard pad infection they reproduced in the laboratory. Where they injected hard pad virus they got a type of reaction and where they then injected egg-adapted virus no case of clinical hard pad disease occurred. His experience was limited to the occurrence of the disease that they called hard pad disease and from which they isolated a virus which they called hard pad virus.

Mr. Hudson said that the inoculation of egg-adapted virus might have a non-specific effect in the same way as the inoculation of horse serum or condensed milk was said to influence the course of the disease.

Dr. Montgomerie said that he would accept that as a possibility. The early observations in using virus as a therapeutic measure in hard pad disease were done with virus from ferret tissue. Later observations were with infected dog spleen emulsion, and Mr. MacIntyre's findings were outstanding. Reports were received from up and down the country, and of 200 cases, 54 were distemper immune, in that the veterinary surgeon had used one or other of the methods of immunisation previously employed. In 44 cases there was no evidence of encephalitis at the time of the injection of the therapeutic virus, and every one recovered. In 10 cases where there was evidence of encephalitis, six recovered and four died. On the other hand, the treatment of 146 dogs believed not immunised was reported. Of the 146 dogs, 48 showed symptoms of encephalitis, 30 died and 18 recovered. Of the other 98 cases of hard pad disease not showing encephalitis and injected with virulent virus, 6 died and 92 recovered. The over-all recovery of 80 per cent. seemed good; the figures for distemper immune animals was 92 per cent. and for distemper non-immune was 75 per cent. That was the result in the trials Mr. MacIntyre ran in co-operation with practitioners, and he would like to acknowledge that co-operation now. On the figures, they could not avoid the conclusion that this was a true bill.

Mr. Roper said that he injected egg-adapted virus in a dog exhibiting signs of being a carrier of Rubarth's disease, and the dog died. It was confirmed later by liver examination.

Dr. Montgomerie said that Baker's work at Cornell showed there was a special danger in trying to immunise a dog which was a carrier of Rubarth's disease. They had not enough evidence to say that they could fully confirm it. He suspected that a dog infected with this virus, not showing the disease, was a bad risk in immunisation. They had known dogs suspected of being carriers of Rubarth's disease going down with that disease after the use of egg-adapted virus.

Mr. Pool asked if there was any theoretical explanation why the virus had a curative effect.

Dr. Montgomerie said there was no theoretical explanation; one found it almost difficult to believe it was a true bill. His difficulty in accepting this was that he did not know the theory; he was far from satisfied it was an interference phenomenon.

Question No. 2

It is not uncommon to see a young dog exhibit symptoms referable to liver and vascular system which seems typical of canine virus hepatitis; recovery follows the usual course but two to four weeks later the dog shows evidence of late hard pad symptoms. Does the forum consider that the initial diagnosis was wrong, or that in fact intercurrent infection, with both viruses exists, with the hepatitis syndrome dominant and masking hard pad symptoms in the early stages? How common is dual infection?

Dr. Larin said that there were some points in this question which were difficult to answer. He doubted whether one could diagnose with reliability either the vascular or liver disturbances in the living dog. There were cases of young dogs exhibiting certain signs of infection, but it could only be suspected that it was either canine virus hepatitis or anything else. The second difficulty was the suggestion that two to four weeks after recovery the dog usually exhibited " symptoms " of late " hard pad " disease. He would suggest the use of the word " signs " instead of " symptoms " as the latter usually meant complaints on the part of the patient. He also thought that it should be difficult to say what " Hard Pad " was even if affected dogs were able to tell of symptoms.

The authors of the first paper on hard pad disease, Dr. Montgomerie and his colleagues, described in their excellent paper, under the name of hard pad, canine encephalitis. Other authors described hard pad disease in rather a different way and it seemed that all these descriptions did not coincide. Thus hard pad disease was never defined as a separate entity. He also had grave doubts regarding the suggestion that hard pad virus existed as a separate entity. Many forms of distemper had been described and thus it was difficult to know which form was called " typical " distemper. Some of these forms probably emerged as a result of infection with different types of the virus. The others might be due to dual or secondary infection with other virus or bacteria.

He also said that not so long ago, canine virus hepatitis was diagnosed as distemper, and there was a possibility of infections in dogs with hitherto unrecognised viruses. He mentioned all this in order to stress the fact that the clinical methods of diagnosis in canine medicine were not perfect enough to rely upon, and to be sure that it was not uncommon to see a young dog exhibiting signs which seemed typical of canine virus hepatitis and, two to four weeks later, after recovery, to show evidence of late hard pad signs.

The next point was on dual infection with canine hepatitis and distemper viruses. That was common because canine virus hepatitis infection was very widespread; out of 100 dogs serologically examined, one would probably find evidence that 99 had the infection. Although the majority of infected dogs usually suffered from a mild or inapparent (subclinical) form of infection with hepatitis virus, it sometimes produced considerable tissue damage. Thus, fresh infection with another virus or bacteria might prove to be fatal. If canine virus hepatitis was widespread, then dual infection with distemper and hepatitis viruses was common. He did not know whether dual infection might differ from case to case in the clinical picture, but he thought that in many cases it would make no difference to the prognosis, which was bad, because both viruses had an affinity to the vascular system.

Dr. Mansi said they must think in relation to hepatitis and to the distemper complex, including hard pad. They could have both virus infections at a time, or have distemper and recover and then hepatitis and die, or vice versa. There was no real difficulty in confirming a dual infection. He did not agree that dual infection was so common and that 99 per cent. of the dogs were infected. It must refer to dogs of certain ages. Dogs reared at Weybridge under three months of age were usually free from infection, but from three months onwards they were susceptible, specially between three and six months.

Dr. Montgomerie said that he would accept that a large number of dogs were infected with hepatitis virus, but it was another question whether they could take Dr. Larin's tests as positive results that the dogs were infected or not. He did not care whether they called the particular disease hard pad, hard pad distemper, part of the distemper complex or not, so long as they knew what they were talking about. If they went on multiplying names they would get confusion. He was sorry hard pad disease slipped out in the profession and in literature; it was not a good name but when they were talking of hard pad disease they knew what they meant. It was interesting to go back in the literature and realise that there were many articles in American journals of the past describing " distemper," not as accepted in Britain, but what they now accepted as a typical hard pad disease. At that time, they in Britain reserved the word distemper for the disease Laidlaw and Duncan described and investigated; the old-fashioned distemper, from which they could isolate a virus giving a typical distemper diphasic temperature reaction as described by Laidlaw and Duncan.

Capt. McBee quoted Baker's work and said it was common for dogs to have hepatitis and for it to go unrecognised, but the question was which came first, hepatitis or distemper. If the dog had distemper first and was then in contact with hepatitis, they could have a problem. The dog went to pieces quickly. If the reverse was true and it had hepatitis first and was then in contact with distemper virus, it did not have the complications.

Mr. Woodrow said that he agreed with Dr. Montgomerie; the clinical picture of distemper to-day was a different entity. There was an excellent summary in THE VETERINARY RECORD a few weeks ago. What they recognised clinically to-day was distinct from the old type of distemper, with a long and ill-defined incubation period, injection of the sclera, persistent diarrhoea, and a temperature which remained at 103° throughout and 78 per cent. of the dogs developed encephalitis. With hard pad the public should realise they were up against something more serious than " corns " and must be prepared for such a high incidence of encephalitis.

Mr. R. S. F. Campbell said that he must disagree with Dr. Montgomerie and Mr. Woodrow on a number of points. Dr. Larin had spoken of mild hepatitis associated with serious tissue damage, and he would like him to explain the term " mild hepatitis "; was it a dog with a slight virus infection or a dog showing few clinical symptoms?

Dr. Larin said he meant a dog showing a few clinical signs of the disease.

Mr. I. M. Lauder said he would like to comment on what Mr. Woodrow had said. His cases with hardened pads seemed clinically to be different to what Mr. Woodrow had found. Much had been said about " old-time " distemper but previous descriptions of the symptoms were not very full. Laidlaw and Dunkin's was remarkably useful work but their clinical account of affected dogs was scanty. The " old-fashioned " distemper was not something clearly apart from hard pad. Mr. Woodrow mentioned some signs of hard pad, but he did not believe these were definite enough to distinguish an entity apart from distemper.

Dr. Montgomerie said he could remember a discussion at the Central when the description was most adequate; quite apart from Laidlaw and Dunkin. In the discussion there was a full description of distemper

as he used to know it; it was different from what they knew to-day in the so-called hard pad disease.

Mr. Bateman said the old-fashioned distemper was not occurring nowadays. He had known dogs immune to distemper which had recovered and been left without the loss of co-ordination. If they were sent to the laboratory, would they be immune or not to hard pad disease? If they injected hard pad virus into dogs 100 per cent. immune to distemper, they would be affected three or four days after injection and they would say it was due to injection of hard pad virus. Knowing as he knew they were immune, the question was whether the dogs at some date were affected by natural infection of another virus or the infection from the laboratory virus.

Dr. Mansi said that immune dogs, if challenged in the laboratory, may show a thermal reaction but no clinical disturbance at all.

Mr. Bateman said the dogs did not react physically to natural infection but would react to the injected virus. They did not react thermally to natural infection.

Mr. Pool asked if a separate encephalitis virus occurred in dogs.

Dr. Montgomerie said there was no doubt they had encephalitis.

Mr. Pool asked whether it was safe to assume that these cases of encephalitis were due to distemper virus or a separate virus.

Dr. Montgomerie said they had published what they believed; that the lesions of encephalitis were due to distemper virus or due to hard pad virus or other things. Rightly or wrongly they believed they could histologically diagnose encephalitis due to so-called hard pad virus. They had not proved themselves wrong yet, it was up to somebody else to prove they were wrong.

Mr. Campbell said that his studies were on 250 dogs and the findings were based on histological data. In the great majority of encephalitic cases, they had recently had distemper infection. Had one taken account of every specific factor capable of producing encephalitis? The matter of encephalitis was a complex one and he was not prepared on the basis of his work to recognise there were as many factors capable of producing encephalitis as Dr. Montgomerie had defined.

Question No. 3

What evidence is there of a true carrier state in canine virus hepatitis? How long, in the opinion of the forum, should a recovered case be isolated? And in cases of outbreaks occurring in a kennels, how long should elapse before stock should be sold or shown?

Dr. Mansi said this was a very delicate question as they had heard that the disease was widespread and he did not know the real danger of carriers among recovered dogs. At Weybridge they had done some experiments with dogs naturally infected and with some experimentally infected. They had failed to find any specific antigen or virus in the bodies of dogs after 41 days from primary infection, although it had been reported that the virus had been isolated from urine after 272 days. That was a single case and it had not been repeated. They had some dogs which were experimentally infected four years ago and they were still showing strong antibodies: they were bitches producing litters and there was no serological evidence that the puppies were immune from the time they were born. When they reached the age of three months, they were found on inoculation to be susceptible to hepatitis. There was no evidence that healthy, susceptible dogs became infected from these other dogs in the kennels. Recovered dogs remained potentially infectious to susceptible dogs for a period up to 15 days only. They had exposed susceptible puppies to possible infection from dogs which had been recovered from the disease for periods varying from 30 to 80 days. None of these puppies so exposed, contracted the disease and when challenged they showed no immunity.

The question asked how long should elapse before stock should be sold or shown. Having had an outbreak in a kennel they should isolate recovered dogs for at least a month, while taking into consideration the recognised rules of disinfecting and then regard the situation as no longer dangerous.

Dr. Larin said that he had observed canine virus hepatitis in breeding kennels for five years and a large proportion of the naturally infected dogs had developed the sub-clinical form of the disease. He stored the virus, in the freeze-dried state, for five years in sealed bottles, and after that period of time three puppies were inoculated with the virus. All three animals developed the fatal form of canine virus hepatitis. The virus was also stable to the heating at 56° C. for 30 minutes.

Dr. Montgomerie said that frankly he did not know how any breeder who knew that hepatitis had occurred in the kennels could offer any guarantee in sending away a dog. He accepted Dr. Mansi's evidence of the period in his experiments but Dr. Larin suggested that his attempts to clean up a kennel had ended in failure. He could not see how a commercial kennel having hepatitis could ever feel satisfied it was not selling a dog infected with or carrying hepatitis virus. Despite efforts, he had failed to eradicate the disease from kennels and he felt little hope for the commercial breeder to eliminate the disease. When was it safe? As far as he could see, next door to never.

Mr. Woodrow said he was satisfied to leave the question to the laboratory men.

Mr. Bateman asked for some enlightenment on the young puppy from an infected kennel. How long was it in good health; until three months later?

Dr. Mansi said experiments showed that from the time the puppies were born until 10 to 12 weeks old, they were in a state of resistance, but this was not due to antibody immunity. If they inoculated puppies when less than eight to 10 weeks old with hepatitis virus, they did not react clinically or serologically; some when reinoculated at about the age of 80 days, became infected. There was no virus or antigen which could be demonstrated in puppies infected with hepatitis virus, when they were less than 10 weeks old, and killed 24 hours or a week afterwards.

Mr. Bateman said that if the puppy was safe at that age, it was at that age it should be vaccinated against distemper and that was why egg-adapted virus should be used as soon as the puppy was two months old.

Dr. Montgomerie said it only related to puppies from immune mothers.

Dr. Mansi—Non-immune mothers.

Dr. Montgomerie—In puppies under eight weeks old from non-immune mothers.

Dr. Larin said that they could argue the matter for a long time because there was not much knowledge of it. During the last five years he was studying virus hepatitis in breeding kennels with the endemic infection, the majority of the dogs developed inapparent (sub-clinical) infections which were detectable only with the aid of the complement fixation test, and those dogs could be considered as carriers of the active virus. Apart from this there were dogs affected with the acute form of the disease which harboured the virus in the tissues for a long time after clinical recovery. Hence the presence of carriers in kennels with endemic virus hepatitis could be considered established. The way of spreading infection, however, still remained unknown. There were litters in which some puppies died of the disease while the others were susceptible to experimental infection with the virus.

Dr. Mansi asked how he knew there were carriers.

Dr. Larin said he thought it was clear. It was known by observations and serological tests made over five years in the breeding kennels where virus hepatitis was endemic. Apart from this the virus was isolated from some dogs a few months after clinical recovery. Therefore, he knew that many dogs in the compound harboured the active virus, but, he did not know, of course, the number of dogs which were shedding the virus around.

Mr. Bateman said that since the beginning of September, he had vaccinated 47 dogs at two months old without any untoward signs.

Question No. 4

(4*a*) *What in the opinion of the forum is the relative value of the egg-adapted vaccine versus killed vaccine followed by virulent virus or the virus/serum methods of distemper prophylaxis?*

(4*b*) *What form of immunisation against* (*a*) *the distemper complex, and* (*b*) *canine virus hepatitis, are common in the U.S.A.?*

Mr. Woodrow said that they had now been using egg-adapted vaccine for 15 months but it was still too soon to judge the duration of immunity. In the 15 months he had hoped to get some idea of the level of immunity they got with egg-adapted virus and to relate this immunity with an analysis of the different types of cases injected and comparisons between those cases at the time of inoculation. In the laboratory they knew that egg-adapted virus protected susceptible puppies otherwise the batches of fresh virus would not be issued. The puppies were immunised under ideal conditions and were free from outside infection; they were different to the street dogs they had to deal with. He must emphasise the good conditions at the time of inoculation. He would like to give figures regarding an outbreak of distemper complex last winter in a foxhound kennels. In December, 1953, 20 couples had the typical symptoms of hard pad distemper. At the same time there were in the kennels 14½ couples which had been inoculated. Only one of these developed hard pad and made a good recovery.

A third type of case concerned dogs bought from dog shops. He persuaded the management of one shop to inoculate all puppies immediately they arrived from breeders with egg-adapted virus. He had inoculated 138 puppies and up to the moment no complaint had been received of any illness in the months following purchase. The animals were from small breeders, were not exposed to infection and were inoculated when reasonably clear of infection. In other dog shops the conditions were different; egg-adapted virus was not given and the purchaser was advised to take them to a veterinary surgeon for inoculation. In this group there were a large number of breakdowns. The lack of immunity in these cases was explained by the fact that the shops were continuously infected and the puppies often showed symptoms of the distemper complex within six weeks of having egg-adapted virus. Other cases were more disturbing; he might immunise a dog, apparently successfully, and it went down with symptoms of hard pad or distemper five or six months later. Some of the cases failed because of the conditions appertaining at the time but there were other factors which could interfere with the development of immunity. He thought that it was complete failure to develop immunity rather than any so-called "breakdown." They required some sort of criterion to know whether a dog had taken, otherwise they did not know whether they had got any immunity at all. He thought that all they could do at the present to ensure the success of egg-adapted virus immunisation was to try to attain proper conditions at the time, to maintain the viability of the virus, see that there was proper sterilisation, and then the only precaution against failure was in the selection of cases. They should refuse to inoculate those doubtful puppies with a doubtful background; if they gave them anything, then give serum.

Capt. McBee said methods of immunising in the U.S.A. were very similar to those in England. The most popular was egg-adapted vaccine, there was enough evidence now to show that it was safe. Compared to the tissue vaccine it was superior; tissue vaccine built up antibodies for a short-lived immunity unless challenged with virus in the field. Most of the time they were building up a short-lived immunity and expected the animal to be in contact with virus to give it life-long immunity. At one time that was the only method; the use of tissue vaccine followed by live virus. There was always the question of whether they were going to get into trouble by the use of the live virus, and whether or not the virus would be transmitted to other animals. The immunisation of young puppies was certainly very popular; they knew they would have no immunity unless they got it from the colostrum of the bitch. If the puppy was from

an immune bitch, it would have enough of the colostrum to keep it immune for six weeks, so there was no doubt of some degree of immunisation at an early age. If there was any question of the bitch's immunity, they should start at six weeks and inject a serum up to three months at which age they could safely give egg-adapted virus and expect a reasonably long-lived immunity. Canine virus hepatitis had been a problem in the United States. At the present time a dual vaccine was being used experimentally. It would be on the market soon; it was very effective.

Dr. Mansi said that they had heard of the possibility of dual infection, that hepatitis was widespread and that egg-adapted vaccine was good. As they had dual infection, why not apply dual vaccination? They had just heard that non-living hepatitis virus failed to give satisfactory results. While the injection of the live hepatitis virus under the skin produced serious if not fatal results, the instillation of this virus in the conjunctival sac of the dog produced a local " eye reaction " accompanied by a very mild infection and ended in complete recovery by the 10th to 13th day after vaccination. The dog may be inoculated at the same time with distemper avianized vaccine. Over 100 dogs had been vaccinated by this method without any mortality. Many of these dogs had been challenged by the inoculation of a mixture of virulent distemper and hepatitis viruses to which they resisted, if inoculated one week or more from the time of vaccination. The only disadvantage was that vaccinated dogs might infect susceptible contacts up to 15 days after vaccination.

Dr. Larin said that the relative immunising value of any vaccine depended upon whether it was made with strains representing the current types of viruses operating in the field. The evidence strongly suggested that in the case of distemper they did not have a homogeneous virus with distinctive characteristics, but, probably, several types. If this evidence was accepted then could much credit be given to a vaccine prepared from one strain only?

The evidence of the immunity produced by egg-adapted virus was controversial. The data regarding the lack of immunity were very strong since there were numerous cases where lack of immunity could not be attributed to bad batches of the virus. In his opinion dogs immunised with the egg-adapted virus might be immune against the type of virus similar to that used for immunising, but where the dogs were infected with a different type of virus there might be a lack of immunity, and that was the present position.

Dr. Montgomerie said he would like to see the evidence that the egg-adapted strain did not immunise ferrets, the most sensitive animals to distemper. In the early stages, experiments had been carried out to determine whether there were different strains. He did not know the evidence which would convince him that there were these strains which were so immunologically distinct.

Dr. Larin said he could not agree with the comparison of the active immunity to distemper virus in the ferret and in the dog. The immunity in the ferret was one thing, and that in the dog was another. He did not think that he could produce evidence yet regarding the egg-adapted virus; that would probably come later. The ferret, as a laboratory animal, was not equally susceptible to all types of distemper virus, and he could prove that by the results of his experiments with 55 strains isolated from material sent to him by veterinary surgeons in practice.

Dr. Montgomerie said that if they had handled distemper in ferrets and in dogs through the years, they would realise as Baker and others had done that the ferret was very vulnerable in distemper immunisation He would 10 times sooner recognise distemper in ferrets than in the dog clinically. If they depended on the dog for experiments they would be in trouble; he had never been out of trouble from the day he had started working on distemper! On the question of failures of immunity, he had not seen the evidence to make him accept the view that the failures were due to virus differences. The strain they were using was a strain derived from a hard pad type; there was no evidence so far of batch failures. That was related to the question of the level of the dose. He had never been prepared to maintain that every vial in the batch was a counterpart of its fellow. There was no evidence that there had been failure to immunise due to failure to produce batches of vials which were not comparable. Failure in the field might be due to the use of antiseptics in sterilising the syringe; and to lack of keeping quality. In the laboratory they stored the vaccine under reasonable conditions and they tested the length of the keeping quality. Lack of response in the animals also let them down. By and large it was remarkable how low the incidence of failure was. He was not in disagreement with Mr. Bateman that the best age for immunisation was two months. They had still got to confirm that with egg-adapted virus; the position was something similar to when they knew immunity was dependent on the injection of a living virulent virus. They were satisfied that under 12 weeks an attempt to immunise might be interfered with by the so-called maternal immunity. He could not be satisfied that immunisation at eight weeks was a perfectly sound procedure on general principle; there might be interference. Reinjection with egg-adapted virus must be sound procedure, if inoculated at a young age.

Mr. Bateman said that they must not loose sight of the fact that the dogs were then let loose, they stood the chance of natural infection. Mr. Woodrow was right when he suggested there was no yardstick; he believed that without a thermal reaction it was difficult to know whether a dog had " taken."

Dr. Montgomerie said that exposure to natural infection was one of the best immunising agents they had. He agreed with Mr. Bateman that immunisation at an early age and the relative early exposure to infection was sound procedure; Mr. Bateman had got away with some things in the past but he took risks to reinforce the immunity he had to get.

Mr. Henderson asked if immunity following egg-adapted virus depended on the multiplication of the virus in the dog.

Dr. Montgomerie said he could not answer that. There might be multiplication to get the level of

immunity. They might be able to answer that in a year's time; but no one could give a satisfactory answer.

Mr. Bushman asked how long the immunity would last with puppies inoculated at eight weeks and how soon with a straight virus to produce a lasting immunity.

Dr. Montgomerie said he could not answer that question.

Dr. Mansi said about three months.

Mr. Halpin asked whether a comparison could be given between the older methods of vaccination and with egg-adapted virus.

Mr. Woodrow said that there were two questions of safety and level immunity. Vaccine virus had produced a good level of immunity over the years but at the expense of some risk. It was still too early to judge the level and duration of the immunity conferred by egg-adapted vaccine.

Mr. O. Jones asked whether it was assumed that all types of dogs had the same immunological picture. Recently he imported some dingoes from Australia; they were captured wild and presumably had not been in contact with domestic infection. The animals were vaccinated with egg-adapted vaccine and all took a disease which was diagnosed on *post mortem* as being a hard pad infection. They came from a wild plain with no natural infection; was their immunity different to that of the domesticated dog?

Dr. Montgomerie said he did not know.

Dr. Mansi—How old were they?

Mr. O. Jones said they were puppies; there were 17 animals and one survived.

Miss Freak asked how long it occurred after inoculation and whether they got the infection in London.

Mr. O. Jones said he would suggest that they got the infection in London. They were inoculated in quarantine. About six weeks later when they were on exhibition, one of them escaped and had an opportunity of getting infected before recapture. Within a month the trouble started and in three or four weeks they lost most of them.

Mr. Woodrow said the same thing occurred in 1949 after vaccine-virus inoculation of a pack of huskies. they were inoculated by the ship's doctor with vaccine sent to South America, followed by virulent virus at Las Palmas. They went into quarantine and went down five weeks later with hard pad traced to horse meat infected at the slaughter-house where a bloodhound was found to be affected with hard pad disease. There seemed to be some difficulty to immunise the miniature poodle. This might be a breed incidence but again an analysis of cases showed that one third of the dogs inoculated by me during the last year were miniature poodles.

Miss Joshua said they should know the background of the puppies they immunised. They should only immunise with egg-adapted virus when they knew the puppies had been isolated for three weeks. She had only met one disaster with egg-adapted virus and was at a loss to account for it. She had seen the puppy six weeks after immunisation when it was in the last stage of encephalitis of the hard pad type. It was aged between seven and eight months. She was convinced that if puppies were immunised at the time they were exposed to infection, the egg-adapted virus masked the physical symptoms for a period up to six weeks; they only saw the results when the dog was in the last stages of nervous syndrome. They should be choosey about puppies and refuse to immunise those whose background they did not know.

Miss Uvarov said it was up to them to take precautions, but they also needed a pointer on the type of puppy not to inject. She would like to know if the forum were in a position to offer them more about the mechanisms of immunity with distemper virus or was the mechanism as obscure as it was in other diseases? Had all the members agreed that there was plurality of distemper virus or only one?

Dr. Montgomerie said that he did not pretend to know the mechanism of immunity. What were they after in egg-adapted virus? Were they after the ordinary immunity demonstrating distemper antibodies in the serum in the injected animal? He felt certain that there were some other levels of immunity at work to resist the virus infection. There must be a plurality of any virus from the virulence point of view; most viruses were not stable and tended to alter here and there. If they were talking of plurality from the immunological point of view, the position was different. As far as demonstrating the difference between hard pad virus and what they defined the true distemper virus, there was no way of defining the immunological difference at the moment. Dr. Larin was convinced there was a plurality of virus, which changed all over the place.

Dr. Larin said he did not know whether distemper virus could stand on its head or its tail. He believed that viruses had neither of these. The ferret was the only laboratory animal in which the virus could be isolated, and had Laidlaw and Dunkin not introduced the ferret as the test host, it would be difficult, almost impossible, to prove, or disprove, the presence of the virus in many cases. The disease induced by the virus in the ferret certainly might deviate, but only within certain limits, and not to such an extent that one would say that distemper virus could produce anything in the ferret. If virus produced almost any disease in a test host, then it was either a virus mixture being dealt with, or the virus was not homogeneous and consisted of several types.

Laidlaw and Dunkin did not say that distemper virus could produce anything in the ferret. They gave an excellent description of the behaviour in the ferret of distemper virus type which was the dominating type at that time. Time had changed since then, and the virus had probably changed also. The virus type described by Laidlaw and Dunkin could be seen at present in the minority of cases only, whilst the other types were in the majority. Since little was known about the subject of active immunity to distemper, the knowledge on this subject was largely empiric. It was known that in virus diseases such as poxes, etc., viremia was an essential part of the illness and a staunch immunity developed after recovery.

In other virus diseases including influenza and common cold virus, invasion of the blood-stream was not an essential part of the disease, and it was commonly agreed that a permanent immunity did not exist in these virus infections. But what of distemper? In so-called " Laidlaw and Dunkin " or " classical " distemper there was evidence that a high fever was an essential part of the disease and this was not so in many cases of distemper which they could see now.

Dr. Montgomerie said the ferret did not live.

Dr. Larin again stressed the fact that the evidence of Laidlaw and Dunkin was that distemper virus produced in the ferret, at that time, a disease which was characteristic, easily recognisable, and could be compared in all essentials with that in the dog. If they accepted that distemper virus produced anything in the ferret, it would be contradictory to the evidence of Laidlaw and Dunkin. At present the majority of current distemper virus strains produced in the ferret are diseases of a different type. So it seemed too regarding the dog, and all this was strong evidence for the existence of different types of the virus.

Dr. Montgomerie said distemper was diagnosed by ferret injection in the laboratory. No distemper virus could have been injected if the ferret lived and when later challenged with known distemper virus, died. Very few ferrets were naturally immune and no ferrets inoculated with the true distemper virus remained alive to be challenged with another virus.

Dr. Mansi said results obtained by vaccinating ferrets with formalised vaccines were conflicting. Ferrets were immunised with vaccine prepared from one strain of virus and then challenged with the homologous strain, if they survived they were challenged with other strains of the distemper complex. No immunological differences between strains were found.

Question No. 5

Occasional cases are seen in which clinical signs of vitamin K deficiency occur during or immediately after an attack of hepatitis. Does the forum feel this possibility is sufficiently important to warrant the routine use of vitamin K or its analogue in treating all hepatitis cases?

Dr. Montgomerie said it was difficult to test vitamin K deficiency; they could not get it from virus infection tests. They must not be confused with the appearance they got from a vitamin K deficiency as the evidence suggested, for vitamin K deficiency was not so sound as it first appeared.

Dr. Mansi said it was suggested that the use of vitamin K might overcome the trouble but he did not think it would do any good as the damage was already in the liver itself. The liver lost its function completely.

Miss Freak said it did appear that vitamin K deficiency was not concerned with hepatitis though there might be symptoms to suggest it.

Question No. 6

What in the opinion of the forum is the connection, if any, between the virus diseases and the occurrence of uterine inertia in the bitch, low foetal vitality, or fading puppies.

Mr. Woodrow said that it seemed the view of the panel that it was due to the presence of canine hepatitis virus and yet Dr. Mansi said that puppies up to three months old were almost impossible to infect. There was no correlation between the two views.

Dr. Larin said that low foetal vitality and fading puppies were probably caused by many factors. They inoculated suckling puppies with canine hepatitis virus and the puppies faded away and died. In his opinion canine virus hepatitis was one of the many factors of high neonatal mortality.

Dr. Mansi said that all puppies received by him that had died after or shortly after birth or to within a few days of birth, were due to causes other than distemper or hepatitis as shown by an examination of over 100 cases serologically. Litter mates which survived were found to be susceptible when they had reached the age of about three months.

Dr. Larin said he had inoculated suckling puppies with canine hepatitis virus and a difference was then observed compared with infected adult dogs. This was that inclusion bodies were absent in the great majority of the suckling puppies infected with the disease under natural or experimental conditions. He knew of only two cases where the inclusion bodies were seen. The first was one of his own cases and the second was a case of his colleague, Mr. Skulski.

Dr. Montgomerie said their kennels had trouble from fading puppies. He was not convinced that it was associated with hepatitis or distemper infection. He had tried to find out but a fading puppy was a fading puppy and he was not convinced it was due to any virus infection.

Mr. Halpin asked about the uterine inertia part of the question.

Mr. Bateman said he had no evidence that hepatitis was related to uterine inertia and he was certain that it was nothing to do with distemper. He found he was able to prevent it by feeding the bitch on a low level of a sulphonamide; all bitches in whelp were treated with it.

Dr. Mansi said he was convinced it was nothing to do with the viruses at all.

Mr. Pay said that he had seen a number of cases where on histological examination there was widespread damage in puppies about a week old. He had searched in the field unsuccessfully, although in one case he found inclusion bodies in a puppy two or three days old.

Question No. 7

Has the forum any evidence that clinical cholecystitis can occur in dogs recovered from canine-virus hepatitis? Some cases recover satisfactorily but show abdominal pain some one to two hours

after food and some evidence of bile suppression. One case of a ruptured gall-bladder had been seen in, what was in retrospect, a fairly obvious hepatitis case; this suggests that inflammatory changes can follow the oedema which we know occurs, is this so?

Dr. Montgomerie said they had little knowledge of what happened in those that died but in those that recovered there must be heavy changes in the liver and in the gall-bladder. If it was anything like that in the *post mortem* then they would feel that it was impossible for the animal to return to normal without some disability to the gall-bladder and the liver.

Dr. Mansi said he had killed and examined many recovered cases and he could not recollect any changes in liver or gall-bladder after 30 days.

Dr. Montgomerie said that clinical cases of hepatitis did recover; it was suggested that there was no marked effect in the gall-bladder.

Mr. Hudson asked whether, in the recovered dog, there was any pathological evidence that it had ever been through hepatitis. There seemed to be conflict of evidence; Dr. Mansi said he had been unable to demonstrate any carrier stage, and that bitches having regular litters of puppies were absolutely normal and that after three months the puppies were susceptible and should be inoculated. How did that fit in with Dr. Larin's experience and that of Dr. Montgomerie. They spoke of the difficulty of getting susceptible puppies. Was it anything to do with the number of animals in the kennels? Was it due, not to a true carrier stage in the virus, but to a carry-over of the infection in the litters? Owing to the small number of breeding bitches Dr. Mansi had eliminated that, because there was no opportunity of a carry-over from one bitch to another; conditions were different in the other kennels. It might be that there was not a true carrier but a carry-over of the infection. Dr. Mansi's results were clear cut. Another point was that the occurrence of what we regarded as typical hepatitis inclusion bodies did not guarantee that the puppy had had virus hepatitis. He had once found typical hepatitis inclusions in the liver of a young pig and yet when the liver was inoculated to puppies and pigs, it failed to produce any infection at all in these animals. That type of inclusion was not absolutely characteristic of the canine hepatitis virus.

Dr. Montgomerie said that there was no doubt in his mind that it was a carry-over rather than a carrier. Against that it was possible to avoid a carry-over stage where protection was adequate. Mr. Hudson had put his finger on an important point by suggesting that in spite of protection, there were opportunities for the virus remaining; the danger of the carry-over must be real. In the ordinary kennels where the precautions were not like those in the laboratories, the carry-over might be a problem. Against that was the experience of the dog that remained a carrier for nine months. It had happened once. Mr. Hudson was quite right in his second point to " except " or " suspect " the so-called typical inclusion bodies being diagnostic of hepatitis.

Dr. Larin agreed with Dr. Montgomerie, and he again stressed the fact that, apart from carriers of latent virus, there were many cases of the active infection without clinical signs. Also, three months after complete clinical recovery following the acute form of virus hepatitis, they proved that the dog still harboured the virus in its organs; evidence in the U.S.A. suggested a longer carrying period. The epidemiology of the infection also suggested the presence of carriers although the stability of the virus to physical factors must be born in mind.

Mr. Woodrow said that in practice in urban areas there was an appalling loss from encephalitis and there was complete inability to treat the encephalitis or to prevent its occurrence. He would like to know if there was any treatment to reduce the incidence of encephalitis. The introduction of mysoline did help. The course was not so severe and the incidence seemed to be lower if it was used as a prophylactic. He would like to know if Mr. Campbell had anything to say on the matter. What were his views on the possibility of the encephalitis, which developed three to six weeks after onset of virus infection, being due rather to demyelination caused by anaphylaxis than to direct invasion of nerve cells by the virus?

Mr. Campbell said he could not relate anything to mysoline at all. He did not know how it might influence encephalitic lesions which developed. On the problem of encephalitis, he said that from a series of histological studies on dogs, where the great majority showed evidence of encephalitis, the lesions seemed to appear three or four weeks after infection as far as they could tell by the comparative picture. On an interim judgment, he was inclined to the view that a high proportion of the dogs infected with distemper virus developed *some* inflammation in the central nervous system; that was a conclusion of histological studies of dogs from *post-mortem* examinations. He could not speak for dogs suffering from distemper which recovered. In those cases where the lesions occurred four or six weeks after infection, associated with demyelination and, looking histologically, due to virus infection, they could nearly always demonstrate inclusion bodies. Nevertheless, he accepted the precaution of Mr. Hudson that inclusion bodies need not necessarily be significant for virus infection. A reaction occurred in some cases of distemper which appeared to develop later than the " viral " encephalitis. They had been able to trace encephalitis in its early and late stages, and in chronic cases much of the early evidences of virus had disappeared. There had been a lot of work in the United States on allergic encephalitis; he did not know whether auto-immunisation with brain tissue might produce canine encephalitis. In the United States it was thought that certain things mitigated the allergic type of lesion and one substance which retarded development was salicylate. There might be some case for the heavy dosage of encephalitic dogs with it. In a survey of distemper, they had sometimes found encephalitis in dogs without nervous

symptoms; they had constantly found histological evidence of an inflammatory change in the brain. In most cases the symptoms of distemper were blatant but he urged practitioners to be careful before stating that there was no distemper infection in a dog.

Dr. Montgomerie asked whether the distemper virus had produced encephalitis in the ferret. In experimental cases with virus, or when they were using a distemper strain produced by them and inoculated that strain, did it produce encephalitis in the dog?

Mr. Campbell said he could not answer that question in relation to the dog.

Dr. Montgomerie asked Mr. Campbell to confirm that he had got a strain of distemper virus that would produce encephalitis in the ferret.

Mr. Campbell said he was not prepared to give any final contribution on it at the present. In certain cases of encephalitis in the ferret seen, the animals had lived for 30 days or more, but in most tests ferrets died 14 days or so after infection; perhaps the ferret did not usually live long enough for the development of the encephalitic lesion. In dogs the inflammatory signs developed about three weeks after infection when they might be impossible to diagnose clinically. Most dogs survived a longer period and a greater number suffered from encephalitis. That was according to the evidence on the number of dogs they had had for *post mortem* at four to six weeks.

Dr. Montgomerie said that one of the stumbling blocks to investigation of distemper was to reproduce the disease as they saw it in the field. They might think they were inefficient because they could not reproduce what they saw. In the hundreds of cases sent, they had tried to breed a strain of virus to produce hard pad and encephalitis and they had gone on through the ferret or directly in dogs and they had nothing like what they had in the field. It puzzled him.

Mr. Bateman said that they did not know to what extent intracranial pressure was increased in dogs with encephalitis. With such dogs much of the pressure was in the lumbar region and it should be punctured if necessary and 10 c.c. of cerebro-spinal fluid taken. He believed it was worth trying in a young dog making no improvement whatever.

Mr. Singleton said that occasionally in practice they could withdraw some of the spinal fluid; he had some cases where there was considerable improvement and one case recovered. In other cases punctures were made every fourth day; up to four were made and there was very little change in condition. Recently he had tried to establish the pressure at which it was held in the normal dog and in encephalitis cases. The work was in its early stages and there was not enough evidence, but the pressure was considerably increased in cases of encephalitis from 4 mm. of water to 18 to 40 mm.

A vote of thanks to the members of the forum concluded the meeting.

Suspected "Blacktongue," Nicotinic Acid Deficiency in a Dog

BY

LIONEL A. HEMSLEY

Croydon

Introduction.—The occurrence of "blacktongue" in the British Isles has recently been mentioned (Edwards, 1955; Swarbrick, 1955). In Scotland the incidence in sheepdogs is estimated at 11 per cent. (Greig, 1953). The case described below occurred in Buckinghamshire. The common factor is a diet of maize meal or porridge oats.

The presence of substantial quantities of maize in the diet is often associated with the production of natural cases of nicotinic acid deficiency in various species, but there is as yet no evidence that oatmeal contains some anti-nicotinic acid factor.

The striking lingual changes commence with marginal areas of infection, followed by papillary atrophy, and even extend to gangrene of the tip (Rhoads & Miller, 1933). These changes are said to be due to lowered tissue resistance, permitting invasion of fuso-spirochaetal organisms (Smith, 1937). A marked febrile reaction results.

Case Report.—The subject was a three-year-old male working sheepdog. A few days previously it had been offered for sale, being valued at £40. The diet fed consisted of porridge with occasional table scraps. Two other dogs on the same diet have never shown clinical symptoms, but this dog has had brief attacks in previous years.

November 5th, 1954.—The dog was depressed, and strings of saliva hung from its mouth. Temperature 103° F. Some buccal ulceration was present, but the most striking symptom was the brick-red tip of the tongue. Compound vitamin tablets were given, pending treatment with nicotinic acid.

November 8th, 1954.—The dog's condition had deteriorated. Temperature 105·5° F. Tablets containing 50 mg. nicotinic acid were supplied, to be given once daily for fourteen days. Parenteral penicillin was administered. The diet had been changed to one containing a high proportion of animal protein.

November 10th, 1954.—The dog was brighter. Temperature 103·8° F. Sulphathiazole tablets were supplied.

Four days later the dog was reported to be much better. After a further week clinical recovery was complete, the dog being sold after a short interval.

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A Method of Serial Blood Sampling and of Injection for Experimental Farm Animals

BY

W. BIANCA

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SERIAL blood sampling over a period of several hours is a common procedure in many investigations.

Most blood sampling involves frequent puncture of the jugular vein which may result in damage to the skin and the vessel. It certainly causes a state of excitement ranging from mild tension to intense struggling depending on the temperament of the animal, the skill of the operator and the instrument used. This excitement is undesirable, particularly in haematological work, since it causes an increase in the concentration of circulating erythrocytes in the blood through splenic discharge. As a result cell counts, haematocrit and haemoglobin values rise. Under severe excitement the rise in cell concentration may become still greater owing to increased fluid filtration from blood to tissue. The level of blood sugar will also increase. The errors introduced by excitement following conventional sampling vary from animal to animal and from sample to sample and are of unknown magnitude. That this may lead to considerable errors can be seen from the following theoretical example. Let the measured total plasma volume of a horse be 40 litres. In estimating the total blood volume let the haematocrit value be in one instance 35 per cent. and in another 40 per cent. The calculated figures for total blood volume will then be 61·5 and 66·7 litres, a difference of more than five litres of blood.

In the course of work on the blood volume of calves

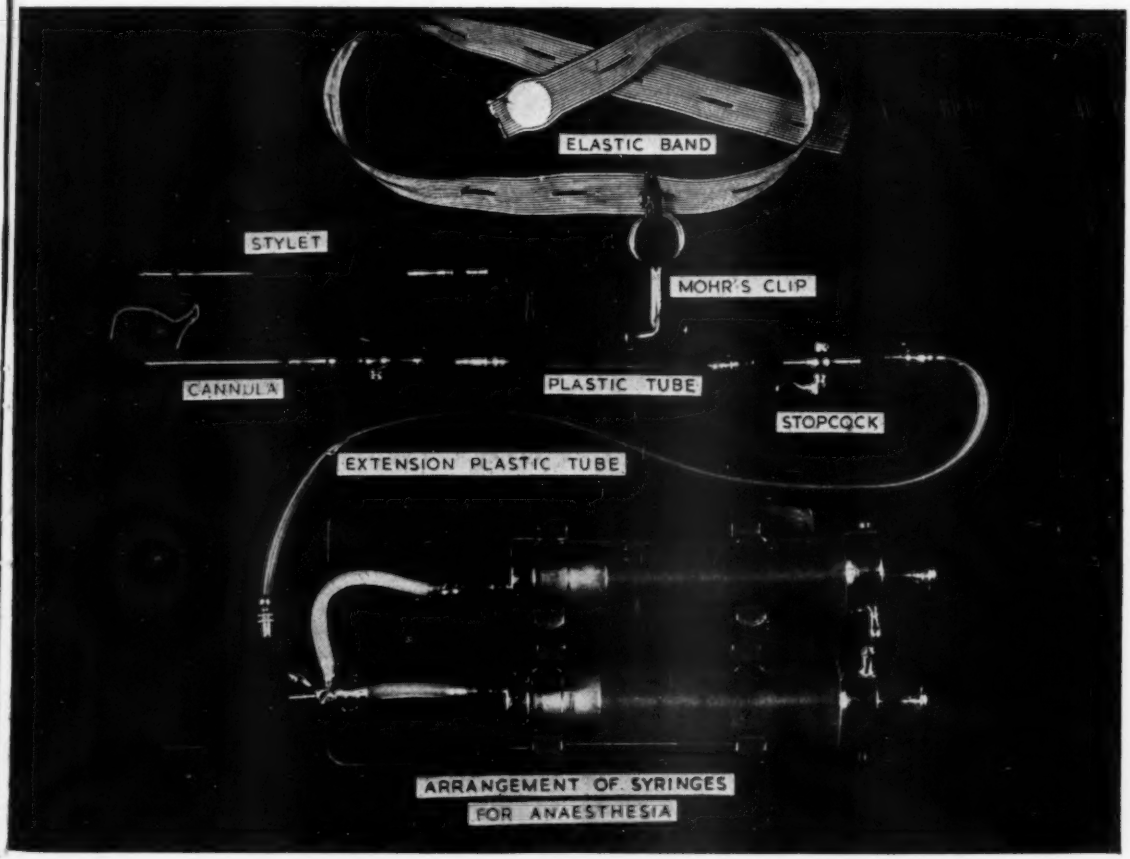

FIG. 1.

it was found essential for the above reasons to devise a method which allowed serial sampling of blood and injection of dye without introducing the undesirable effects just mentioned. The instrument shown in Fig. 1 was found satisfactory. It consists of the following parts. (1) A trocar comprising a stylet and a cannula with a stopcock. (2) A plastic tube with a male adaptor fitting the cannula and a female adaptor fitting a syringe. The tube can be closed with either a Mohr's clip or a stopcock. The clip can be manipulated with the forefinger and thumb of the same hand which holds a sample bottle and allows of quick and quantitative letting of blood. The stopcock is preferentially used when a sample is drawn into a syringe or during injection. The plastic tube provides a flexible connection between the experimenter's hand and the animal. (3) An elastic band with holes in it to keep the trocar in place on the animal's neck and to which the ring of the clip may be hung between samples. (4) Two record syringes mounted on a perspex base and having a joint outlet with a two-way tap for use in intravenous anaesthesia.

The method is as follows. The trocar is inserted into the jugular vein. The stylet is then withdrawn and the flow of blood stopped by turning the stopcock of the trocar cannula. If the skin is very tough it is advisable to perform the puncture in two stages. First the skin is pierced after being gripped between forefinger and thumb of the left hand and pulled taut as in subcutaneous injection. The vein is then entered with a short stab. The plastic tube is attached to the indwelling trocar and the latter is secured to the animal by buttoning the middle of the elastic band to the trocar stopcock and fastening its two ends round the animal's neck. A small amount of blood is now allowed to run out, after which a few millilitres of heparinised saline are injected to prevent clotting in the cannula. Where it is undesirable to introduce any liquid into the blood stream a volume of saline just sufficient to fill the injection pathway is used. The animal is now left until all excitement has ceased which may be checked by taking its pulse rate. Discarding the first few millilitres of liquid from the tube, the first blood sample is then taken. After this the trocar and tube are refilled with heparinised saline and left *in situ* until again required.

The errors due to excitement, already discussed, can be greatly reduced by this method since a suitable time interval can be allowed between the puncture and the collection of the sample. An example of the change occurring in the blood cell percentage can be seen from the table (*top of next column*).

Time after puncture (min.)	Blood sample number	Haematocrit (per cent.)
10	1	34·4
15	2	30·7
25	3	30·3
30	4	30·1
35	5	29·9
40	6	30·2
45	7	30·1
55	8	30·1
65	9	30·1
75	10	30·5

The samples were collected by the method described. The steep decline in haematocrit values between samples one and two suggests that still higher values may have occurred immediately after insertion of the trocar. In this instance virtually stable values were obtained about 20 minutes after puncture.

Apart from offering the advantages of reducing the undesirable effects of excitement and of tissue damage the method has proved useful in allowing accurate timing in serial blood sampling. This is of practical value in all work dealing with the time/concentration relationship of a substance introduced into the circulation. The method has also been used with success for intravenous anaesthesia especially if the anaesthetic is a viscous one. For anaesthetic maintenance over long periods the trocar is fitted with an extension plastic tube which is connected to the joint outlet of the double syringes (Fig. 1). One syringe is filled with anaesthetic, the other with saline for rinsing the injection pathway. Finally the method should be suitable for blood sampling in exercised animals such as horses that are being subjected to a walking test, since the indwelling cannula allows blood to be drawn immediately after exercise has ceased and without introducing errors due to excitement.

Current Literature

DIGEST

Gamma Rays For Fly Control

A novel and ingenious approach to the problem of fly control is described by Bushland, *et al.* (1955), who successfully eliminated the screw worm fly, *Callitroga hominivorax*, from the island of Curacao by the release of sterilised male flies. The female screw worm fly, which causes severe myiasis in livestock, will only mate once, as opposed to the male fly, which will mate repeatedly. It was observed that when a normal female fly was mated with a laboratory-bred sterilised male, she refused further matings and subsequently laid a batch of sterile eggs. It was reasonable to assume, therefore, that the liberation of sterilised male flies would affect a substantial reduction in a natural screw worm fly population. A search was made for an isolated population of the screw worm fly, which would allow the effects of this new method of control to be evaluated. For this purpose the island of Curacao was selected, as it carried a heavy screw worm fly infestation, and it was beyond the flight range of flies from the South American mainland. Flies were cultured artificially and sterilised by exposing the pupae to gamma rays, using cobalt 60 as the source of radiation. Four hundred sterilised male flies were released per square mile of the island, each week. The density of the natural fly population was assessed from the number of egg clusters laid on wounded goats which were distributed throughout the island. At the commence-

ment of the experiment, over 10 egg clusters were laid on each goat weekly. At the end of the fourth week 70 per cent. of the egg clusters were sterile. Four months later, in November, 1954, the last egg mass was laid (which was non-viable), and since this time, no further cases of myiasis due to the screw worm fly have been reported on the island.

D. W. J.

Reference

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ABSTRACTS

Encephalomalacia in Chicks. SINGSEN, E. P., BUNNELL, R. N., MATTERSON, L. D., & KOYEFF, A. (1955). *Poult. Sci.* **34.** 262-271.

The authors state that in the absence of " dietary stress " (such as the presence of fish oil in the diet) young chicks show a relatively low requirement for vitamin E—less than 3 I.U. per lb. of food—and that a low blood level of vitamin E does not, *per se*, result in encephalomalacia.

The addition of a fish oil to the diet resulted in a decrease in the blood tocopherol level and an increase both in the mortality rate and in the incidence of encephalomalacia. This effect was most marked when the diet was low in vitamin E, when the addition of only 1 per cent. of fish oil gave rise to encephalomalacia.

The mortality from encephalomalacia induced by the addition of 2 per cent. of fish oil to the diet was prevented completely when the diet contained 11 I.U. of vitamin E per lb. and the authors suggest that while the vitamin E requirement of the chick cannot be stated at a fixed or specific amount, the presence of 7 to 11 I.U. per lb. is sufficient to prevent the appearance of encephalomalacia under most field conditions. Similarly, this induced mortality was completely prevented by the addition of 0.025 per cent. of diphenyl-P-phenylenediamine (DPPD) to the ration, and it is suggested that 0.0125 per cent. would be effective under most field conditions.

The authors consider that vitamin E prevents encephalomalacia by acting as a general biological antioxidant. They found that the DPPD was effective added to the ration when it was first compounded, added immediately before feeding or injection into the chicks parenterally. They believe that it exerts its effect not by protecting the vitamin E in the ration or in the body, but by acting as a biological antioxidant in a parallel manner to vitamin E.

E. A. G.

Breeding for Resistance to Lymphomatosis. COLES, R. (1955). *Poult. Sci.* **34.** 312-322.

Poultry breeders are often advised that, as a method of combating lymphomatosis, they should breed from hens as opposed to pullets, on the grounds that the offspring of hens are more resistant to the disease.

The author discusses this advice in the light of his own attempt, over a period of five years, to breed a resistant strain of White Leghorns. This attempt was apparently successful, but in the early stages of the experiment it was found that the progeny of second year birds showed a higher mortality from lymphomatosis than had their progeny of the previous year. He suggests that when a resistant line is not strongly established, the ability of a bird to transmit effectively resistance to lymphomatosis may be diminished in her second year, and he therefore queries the wisdom of advising poultry keepers to breed from hens unless a fair measure of genetic resistance already exists in the flock or unless there is only slight exposure to infection.

It was also found that the progeny of dams with high fertility and hatchability rates showed a continued incidence of lymphomatosis, and the author suggests that while breeding for resistance may be successfully accomplished, it may be achieved only at the cost of some loss of fertility and hatchability.

E. A. G.

Ergot Poisoning in Cattle. DRING, J. C. (1955). *St. vet. J. Minist. Agric. Lond.* **10.** 14-18.

The author described an outbreak of ergotism in a herd of milking cows that had access for eight days to a field of rye and mixed grasses heavily ergotised with *Claviceps purpurea*.

The first symptom shown was a fall in the milk yield. This occurred six days after the herd had been turned into this field. Shortly afterwards, 16 of the 43 cows and heifers became lame. This affected chiefly the older, heavier milking cows, and, except in one case, was confined to the hind feet, which showed a cold, hard, insensitive swelling with a sharp line of demarcation about three inches above the fetlock. Sloughing of the affected parts commenced two weeks later. The ears and tails were unaffected. No abortions occurred.

Treatment with sulphamezathine (100 c.c. s/c) did not affect the course of the condition. Only nine of the 16 affected animals recovered, and the remaining seven had to be slaughtered.

Twelve in-pig sows and gilts that grazed the ergotised pasture appeared to be unaffected.

E. A. G.

BOOKS RECEIVED

British Pharmacopoeia Addendum, 1955. Published for the General Medical Council by the Pharmaceutical Press, 17, Bloomsbury Square, London, W.C.1. Price 21s.

Know Your Horse by Lt.-Col. W. S. Codrington, T.D., M.R.C.V.S. (1955). Max Reinhardt Ltd., 66, Chandos Place, London, W.C.2. Price 21s.

ADVERTISERS' ANNOUNCEMENT

BURROUGHS WELLCOME & Co. announce the introduction of "Aerosporin" brand Compound Ointment (Veterinary) for the treatment of infected wounds, and infections of the ear and eye in small-animal practice. The combination of the two antibiotics, polymyxin B and bacitracin in this ointment provides a powerful antibacterial action which covers a wide range of organisms, both gram-positive and gram-negative. The inclusion of benzocaine greatly enhances its use in the treatment of painful conditions of the eye and ear.

"Aerosporin" Compound Ointment is issued in tubes of 10 grammes with special nozzle.

Memoirs

PROFESSOR J. D. STEWART

JAMES Douglas Stewart died at his home in Sydney on September 17th last at the age of 86. He had lived to see the fulfilment of many of his aspirations, in particular the full development of the Sydney Veterinary School, for the founding of which he was responsible.

Born near Sydney, Stewart went to Edinburgh for his veterinary training and took his M.R.C.V.S. at the Royal (Dick) College in 1893. On returning to his native land he became Lecturer in Elementary Veterinary Science at the Sydney Technical College. Later he joined the New South Wales Department of Agriculture and in 1907 became Chief Inspector of stock. These posts were, however, but preparation for his chief mission in life and in 1909 he was elected into the newly established Chair of Veterinary Science in the University of Sydney and set about organising a new veterinary school.

The incorporation of the Veterinary Colleges into Universities, which has taken place during the last few years in this country, was anticipated by Stewart by some 40 years. From the start, the Sydney School was an integral part of the University.

The Sydney Veterinary School

In the early days of the new School, Stewart was confronted with considerable difficulties. The number of students fell to a low ebb and the only other veterinary school in Australia, that in Melbourne, closed and has not been re-opened. Stewart had to face these problems with little support for there were, at that time, only a handful of qualified veterinarians in New South Wales, but he had the necessary determination, character and personality to overcome them. Not only has the Sydney School survived its early vicissitudes but it has achieved a status worthy of a great livestock-producing State. It has been regarded by many as the national School of Australia and has become also the chief centre of veterinary education for New Zealanders.

The School is a material and permanent monument to Stewart's foresight and scientific and professional endeavours, but those of us who knew him well will probably remember him best for his personality; he combined a notable dignity with a very human and kindly nature which endeared him to all his students.

During his life he held many advisory posts and served on many committees, often as chairman. One of the honours he valued most was the Honorary Fellowship of the Royal College of Veterinary Surgeons, to which he was elected in 1936. He was also made an Honorary Associate of the R.C.V.S. in 1949. On his retirement from the Chair in 1939, the Senate elected him Emeritus Professor and he remained remarkably active in mind and body. It must have been a source of much gratification to him, as indeed it is to all his old colleagues, that the family name continues to occupy a prominent rôle in the profession in Australia: recently his son, Douglas Forsyth Stewart, D.V.SC., became Officer-in-Charge of the McMaster Animal Health Laboratory in Sydney.

PROFESSOR D. H. UDALL

Professor D. H. Udall, B.S., D.V.M., D.SC. (HON.) of the Department of Veterinary Medicine at the New York State Veterinary College, Cornell University, died on September 9th, 1955, aged 81 years. Professor Udall was well known to veterinary surgeons in this country as the editor of *The Cornell Veterinarian*, a post which he held from 1918 to 1939, and as the author of one of the standard works on "The Practice of Veterinary Medicine," the sixth edition of which was published last year. In 1937 he was awarded the International Veterinary Congress Prize for his work on bovine mastitis.

Professor Udall was born at Craftesbury, Vermont, on February 9th, 1874, and studied at Vermont and Cornell Universities. He was appointed Associate Professor of Veterinary Surgery at Ohio University in 1903, and in 1908 he became Professor of Veterinary Medicine and Superintendent of the Ambulatory Clinic at the New York State Veterinary College. Since 1942, however, he has been Professor Emeritus at the Cornell University.

MR. A. BLAKE

Mr. A. Blake, F.R.C.V.S., D.V.H., of Nailsworth, Gloucester, died on September 25th, and a special memorial service was held for him at St. Andrew's Church on Sunday, October 9th, at 11 a.m. Mr. Blake was an Alderman and had been a member of the Droitwich Borough Council for 30 years; among those present at the service therefore was the Mayor of Droitwich, together with the Aldermen and Councillors, and a large number of his personal friends.

In his 85th year, Mr. Blake was one of the few remaining veterinarians of his generation who had known Professor Hobday personally. After a short appointment with the Shropshire County Council, Mr. Blake went to Burma as the Chief Veterinary Officer for Rangoon and subsequently became responsible for what was, in effect, the administration of the Veterinary Department for the whole of Burma. He was reputed to have saved Rangoon £1,000,000 in town sanitation alone and so successful was his system that it became the pattern for similar work throughout the country.

Mr. Blake first went to Droitwich in 1926 after retirement from his long and distinguished career in the East, but his enthusiasm for public service remained unquenched and he was twice elected Mayor of the Borough. The Mayor and Councillors paid particular tribute to his work as Chairman of the Finance Committee and to his great work in the field of local government.

News and Comment

OPENING OF THE NEW PFIZER ANTIBIOTICS FERMENTATION PLANT

The new Pfizer antibiotics fermentation plant, which was opened in Sandwich on September 30th, is the first plant to be built outside America and has been financed by the parent organisation, Chas. Pfizer & Co., Inc., of Brooklyn, New York. The plant has been designed to supply the sterling area with terramycin which, until recently, had to be imported from America for processing into various clinical and agricultural forms.

John J. Powers, Jnr., Senior Vice-President of the American company, flew from New York for the opening ceremony and introduced Lord Brabazon of Tara to an audience of more than 500 distinguished guests. Among them was Lady Fleming, widow of Sir Alexander Fleming, and the veterinary profession was officially represented by the President of the Royal College of Veterinary Surgeons, Mr. H. Sumner, together with Dr. W. R. Wooldridge, Senior Vice-President of the Royal College and Chairman of the Parliamentary and Public Relations Committee, B.V.A. Mr. J. L. McGirr represented Dr. A. W. Stableforth, Director of Veterinary Research for the Ministry of Agriculture, and several other veterinary surgeons were there by invitation. Many heads of scientific departments, colleges, and universities were among the guests, and Mr. John J. Powers said:—

"We are gathered here at the commencement of what we hope is the most modern plant of its kind in Europe. It is a British plant, conceived by British brains, installed by British hands, and operated by Britishers. I speak to you to-day as an American because it is an opportunity to put into words a sense of appreciation for the beginning here in this land by a wonderful man of a great era.

"I am referring, of course, to the discovery of penicillin by our dear friend Sir Alexander Fleming, and all that followed. I think that I may presume to speak for the scientific world of America in saying to-day that this is only a small return to English soil of what, one day before, we took from it.

"These times move fast with respect to antibiotics considering the fact that only a decade ago was first brought to our use the wonderful drug, penicillin. It has a range of effectiveness against about 25 pathogenic micro-organisms which cause disease, but by contrast, the drug terramycin, which is one of those which will be manufactured in this plant, has an effectiveness against about 100 pathogenic micro-organisms. Consider the wide range of disease which has been halted.

"I think I might add that terramycin is not just a contribution to the medical world. In over 2,000 papers which have been published by investigators of this drug, many are devoted not only to its medical advantages but to the fact that another new era is open—that of animal nutrition represented by feed supplements made available to the farmers throughout the world."

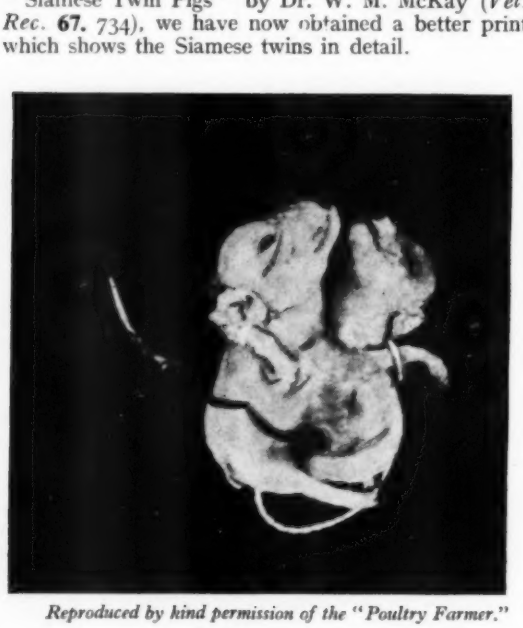
Mr. Powers then called upon Lord Brabazon to dig a sample of soil from the new site and to inaugurate the new building programme. This function having been completed, Lord Brabazon said that he had always had "a fundamental admiration for the fundamental research scientist. It is very different in *ad hoc* research in which you know what you want and get busy chasing it, but in fundamental research you play about with the subject you are interested in and sometimes, not always, there comes a germ of an idea and a discovery, that when developed, changes the history of the world."

H. Sumner, Esq., D.V.H., M.R.C.V.S., President of the Royal College of Veterinary Surgeons, and Mr. R. A. Paterson, M.R.C.V.S., and Mrs. Paterson outside the Hotel Metropole before leaving for the formal opening ceremony of the new Pfizer Antibiotics Plant at Sandwich, Kent.

Mr. J. A. Rodgers, Director and General Manager of Pfizer Limited, concluded the official opening by reminding the gathering that terramycin would no longer cost dollars but would become a British export to doctors and veterinary surgeons in the sterling area. The guests then dispersed to tour the fermentation plant and to visit the exhibition devoted to the History of Chemotherapy and the Discovery of Antibiotics.

SIAMESE TWIN PIGS

In view of the interest aroused by the article on "Siamese Twin Pigs" by Dr. W. M. McKay (*Vet. Rec.* **67.** 734), we have now obtained a better print which shows the Siamese twins in detail.



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ANIMAL WELFARE LEAGUE OF VICTORIA, AUSTRALIA

The assistance of the B.V.A. has been requested in putting the Animal Welfare League of Victoria (Australia) in touch with a veterinary surgeon or veterinary surgeons who may be interested in a permanent and full-time appointment they have to offer.

Further particulars may be obtained from:— The Secretary/Manager, The Lort Smith Hospital, 24, Villiers Street, North Melbourne.

TEST OF COMBINED TRAINING

At the Horse of the Year Show, Harringay, a special Combined Training event, based on the principle of the *Prix Caprilli*, was staged out of doors on Friday, October 7th. Among the competitors was Miss Kathleen Morant, M.R.C.V.S., a graduate of the Royal Veterinary College, London, riding her black horse, Bella. Miss Morant's horse gave proof of a good all-round education by obtaining a clear round in the jumping session which was marred only by the fact that she was debited with five time faults.

APPOINTMENTS IN THE UNIVERSITY OF BRISTOL

The following appointments have recently been made to the staff of the School of Veterinary Science:—

P. M. Biggs, B.SC., M.R.C.V.S., Lecturer in Veterinary Clinical Pathology; M. J. Manners, M.SC. (AGRIC.), N.D.A., Assistant Lecturer in Animal Husbandry; H. Pearson, B.V.SC., M.R.C.V.S., Assistant Lecturer in Veterinary Surgery; and Sheila B. Carter, M.SC., PH.D., Assistant Lecturer in Veterinary Physiology.

MRS. J. G. WRIGHT
A Personal Tribute

Mrs. D. I. Glover writes:—

The tragic news of the sudden death of Mrs. J. G. Wright will come as a profound shock to her many friends. She was well known at Liverpool University and in her own village because of the wide range of her interests. To her family she was the perfect wife and mother, and to those of us who shared her friendship a gay, lovable and truly delightful personality. My own personal close contact over many years has given me the happiest possible memories and that is a consolation I shall treasure. Her family share these memories and to them in their inestimable loss, we, her friends, express very deep and sincere sympathy.

MRS. ALISON BROWN
An Appreciation

Mr. W. Bertram Shaw writes:—

The late Mrs. Alison Brown was a lady in the truest and gentlest sense of the word; she never lost her grace and charm, relics of the Victorian era, even during the moments of stress which occur in a country practice and she carried them to the end. Whilst I have always considered it an honour and a privilege to have seen practice as a student with the late John Brown, it will always be a treasured memory to have met such a personality as the late Mrs. Brown. She was hostess and friend to so many of the stalwarts of the profession of yore that it would be a pity if her passing went unnoticed in the official journal of the profession which meant so much to her.

PERSONAL

Birth

WRIGHT.—On September 23rd, 1955, at Irvine Central Hospital, Ayrshire, to Nessie, wife of E. D. Wright, M.R.C.V.S., Veterinary Department, Nigeria, a daughter, Karen.

Engagement

MCNICOL—CLARK.—The engagement is announced between Ogilvie Duncan McNicol, M.R.C.V.S., 5, Cambridge Avenue, Lincoln, son of the late Duncan and Mrs. I. McNicol (formerly of Mosspark, Glasgow, and now of Stonehaven), and Anne, daughter of Mr. and Mrs. Robert Davey Clark, Houghton le Spring, County Durham. The marriage will take place quietly in Lincoln in November.

Marriage

PACKER—CHAMBERS.—On September 3rd, 1955, at St. Mathew's Church, Pontypridd, Derek O. Packer, M.R.C.V.S., "Rocklands," Bedwlwyn Road, Ystrad Mynach, Glamorgan, to Audrey C. Chambers, "Norwood," Ralph Street, Pontypridd, Glamorgan.

Deaths

Mrs. Alison Brown died at her home in Saltburn Road, Invergordon, at the age of 94 years. As the widow of the late Mr. John S. Brown, M.R.C.V.S., she was well known to many members of the profession.

MACKIE.—Dr. T. J. Mackie, Professor of Bacteriology and Dean of the Faculty of Medicine in Edinburgh University, died at his home on October 6th, 1955, aged 67 years. An appreciation of his life and work will be published in our issue of October 22nd.

R.C.V.S. OBITUARY

We record with regret the deaths of the following members of the profession:—

BLAKE, Arthur, of The Deverells, Nailsworth, Gloucestershire. Graduated London, May 9th, 1893; died September 25th, 1955, aged 84 years.

MITTON, Harry (Capt. late R.A.V.C.), Olicana, Briarwood Avenue, Bury St. Edmunds, Suffolk. Graduated London, December 17th, 1896; died October 4th, 1955, aged 82 years.

COMING EVENTS

October

19th (Wed.). Annual General Meeting of the Northern Division of the Victoria Veterinary Benevolent Fund Ladies Guild in the Northern Hotel, Aberdeen, 2.30 p.m.

Meeting of the Section of Comparative Medicine at the Royal Society of Medicine, 5 p.m.

20th (Thurs.). Ordinary General Meeting of the South-eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

General Meeting of the Herts and Beds Division, B.V.A., at 70, Holywell Hill, St. Albans, 7.30 p.m.

21st (Fri.). General Meeting of the Mid-West Division, B.V.A., at the Berkeley Café, Clifton, 2.30 p.m.

22nd (Sat.). General Meeting of the Society of Women Veterinary Surgeons in the Veterinary Hospital, Pembroke Gardens, Liverpool, 2.30 p.m.

26th (Wed.). Meeting of the R.A.V.C. Division, B.V.A., at the Zoological Society of London, Regent's Park, London, N.W.1, 12 noon.

28th (Fri.). Meeting of the Lincolnshire and District Division, B.V.A., at the Saracen's Head Hotel, Lincoln, 2.15 p.m.

29th (Sat.). Autumn Meeting of the Scottish Metropolitan Division, B.V.A., at the Scottish Milk Marketing Board, Newlands Cattle Breeding Centre, Perth, 2.45 p.m.

First Annual General Meeting of the Veterinary Officers' Section of the Ministry of Agriculture Branch of the Institution of Professional Civil Servants in the Auditorium of the Wellcome Research Institute, 183-193, Euston Road, London, N.W.1, 2 p.m.

November

4th (Fri.). The Annual Dinner and Dance of the Royal Counties Division, B.V.A., at the Chequers Hotel, Newbury, 7.30 p.m.

8th (Tues.). Ordinary General Meeting of the Society of Practising Veterinary Surgeons at the Bell Hotel, Leicester, 2.30 p.m.

R.C.V.S. EXAMINATIONS

November 4th, Friday.—Pharmacology, etc. Examination (Revised Syllabus). Written Examination.

November 7th, Monday.—Pharmacology, etc. Examination (Revised Syllabus). Oral and Practicals (London and Dublin).

November 8th, Tuesday.—Animal Management Examination, Dublin (Probable date).

December 8th, Thursday.—M.R.C.V.S. Written Examinations.

December 9th, Friday.—M.R.C.V.S. Written Examinations.

December 12th, Monday.—M.R.C.V.S. Practical and Orals commence (Panels "A" and "B").

COUNCIL AND COMMITTEE MEETINGS IN LONDON

Wednesday, November 2nd

10.30 a.m.—Veterinary State Medicine Committee.

2.00 p.m.—Parliamentary and Public Relations Committee.

3.15 p.m.—Home Appointments Committee.

Thursday, November 3rd

10.30 a.m.—Organising Committee.

2.00 p.m.—General Purposes and Finance Committee.

Friday, November 4th

10.30 a.m.—Council.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Aberdeen. Cairnbrogie, Udny (Oct. 10).

Cheshire. Carnsdale Farm, Barnston, Wirral (Oct. 3).

Derby. School Farm, Hollington (Oct. 7); Ley Hill Farm, Doveridge (Oct. 8).

Dumfries. Dairy Farm, Fountainbleau (Oct. 8).

Lancs. Shay Farm, Longridge, Preston (Oct. 3).

Leics. Bunkers House Farm, Market Harborough (Oct. 3).

Lincs. Buckberry Farm, Ingoldsby, Grantham (Oct. 4).

Norfolk. Ling Farm, Wortham, Diss (Oct. 7); Manor Farm, Wortham, Diss (Oct. 8).

Perth. Cairnie Farm, Glencarse (Oct. 3).

Salop. New House Farm, Spoonley, Market Drayton (Oct. 7).

Stafford. Copes Drive Farm, Tamworth (Oct. 7); Greyfields Court, Kinver, Stourbridge (Oct. 10).

Wigtown. Clanyard Farm, Dunmore, Stranraer (Oct. 8).

Yorks. Colls Farm, Fellbeck, Pateley Bridge, Harrogate (Oct. 3); Cornborough Manor Farm, Sheriff, Hutton, (Oct. 4); Hunster Grange, Rossington, Doncaster; Lower Brock Well Farm, Triangle, Halifax (Oct. 6); Salutation Farm, Little Fencote, Northallerton (Oct. 10).

Fowl Pest

Essex. Waltham Bury Farm, Great Waltham, Chelmsford (Oct. 3).

Lancs. 1 and 2, Sea View, Marsh Lane, Longton, Preston; Glenfield Poultry Farm, Dale Avenue, Longton, Preston (Oct. 1): Ribblesdale Farm, Hoole, Preston (Oct. 2); Mill Hay Farm, Back Lane, Longton, Preston (Oct. 3); Baldrine Poultry Farm, Nut Lane, Simister, Prestwich, Manchester (Oct. 4); Same Yet, Simister Lane, Simister, Prestwich, Manchester; Moor Hey Farm, Little Hoole, Preston (Oct. 8).

Norfolk. Perry's Allotments, Cobholm, Gt. Yarmouth (Oct. 4); West End Allotments, Cobholm, Gt. Yarmouth (Oct. 6).

Oxford. Home Farm, Weston-on-the-Green, Bicester (Oct. 3).

Swine Fever

Bucks. 128, Hitcham Road, Burnham (Oct. 10).

Dorset. Piggeries, Wheelers Lane, Knighton, Cangord Magna, Poole (Oct. 5); The Piggeries, Manor Farm, Broadway, Weymouth (Oct. 10).

Essex. Felmore Farm, Timperley Lane, Basildon (Sept. 30); Hill Road Piggery, Hill Road, South Benfleet, Essex (Oct. 10).

Flint. Brook Farm, Seabourne Road, Seabourne, Chester (Oct. 11).

Gloucs. Footbridge, Winchcombe, Cheltenham (Oct. 4).

Hants. 121, Park Lane, Kempshott, Basingstoke (Oct. 10).

Herts. The Forge, Widford Ware; Harthall Acres, Harthall Lane, Abbots Langley, Watford (Oct. 4); Crumlin Farm, Flaunden Lane, Bovingdon, Hemel Hempstead (Oct. 10).

Kent. Cladblow Farm, Thurnham (Oct. 11).

Lancs. Alderfield, Clifton Lane, Clifton, Preston (Oct. 7).

Lincs. Edendorke, Church Lane End, Winthorpe, Skegness (Oct. 5).

Norfolk. Whitegates, Southery, Downham Market (Oct. 7).

Somerset. North Street Farm, Stoke-under-Ham, Yeovil (Oct. 10).

Stirling. Firview, The Loan, Linlithgow, West Lothian (Oct. 5).

Sussex. Green Lane Holding, Burgess Hill (Oct. 4).

Warwick. Alma Croft Farm, High Street, Warstock, Birmingham, 14 (Oct. 10).

Westmorland. Keastwick Cottage, Kirkby Lonsdale, Carnforth (Oct. 4).

Wilts. Orchard Farm, Netheravon, Salisbury (Oct. 4).

Yorks. Hammonds Farm, Austerfield, Doncaster (Oct. 5); Redhouse Sanction, York; Piggeries, Moon Lane, Morley (Oct. 11).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

VETERINARY EXAMINATIONS FOR INSURANCE PURPOSES

Sir,—I recently received a request from an Insurance Company to go and examine a horse for insurance purposes which necessitated a journey of 12 miles, the fee for this examination, journey and report offered by them was 12s. I telephoned the insurance office in question saying that there must have been some mistake in that the fee was absurdly low, in fact uneconomic, especially as insurance companies now paid doctors about two guineas for an insurance examination in the consulting room. The office told me that I was expected to make the call " when passing " or whilst " that way." I pointed out that it was dishonest to expect me to make someone else pay for the journey. The insurance company then wrote to me stating that the B.V.A. has agreed to this fee and procedure.

I am informed by the B.V.A. that these fees were arranged with the Tariff Companies in 1945 and, although they were considered unsatisfactory, efforts to get them improved have so far been unsuccesful. Why is it that members of the B.V.A. Council allow themselves to be browbeaten by Insurance Companies? In these days when negotiations fail there is a strike. Why therefore should we examine animals at an uneconomic fee?

Yours faithfully,

G. E. JOSS.

6, Earlswood Road,
Redhill,
Surrey.

October 2nd, 1955.